creased during trials of codeine (up to 120 mg/day) and naltrexone 100 mg/day. However, she became tolerant to these medications and experienced a significant loss in efficacy. Her OCD had been inadequately responsive to treatment with fluoxetine and fluvoxamine. Additionally, fluvoxamine precipitated a mixed manic state, which persisted at her hospital admission despite fluvoxamine discontinuation 2 weeks earlier.

Tramadol 50 mg every 6 hours was initiated, and the patient kept a daily diary to document the onset and duration of therapeutic effect. An immediate (within 15–30 minutes) and dramatic decrease in tics lasting 3.5 to 4 hours was observed by the patient, treating physicians, and nursing staff. Tramadol 100 mg produced approximately 5 hours of marked reduction in tics, but the patient's rebound in tics appeared more robust. At Ms. A's request, tramadol was rescheduled at 50 mg every 4 hours, which produced 3.5 to 4 hours of decreased symptoms and reduced interdose breakthrough symptoms. She reported that her tics usually awakened her during the night, and with tramadol 50 mg every 4 hours, she reported significantly fewer awakenings.

In contrast to previous trials with codeine and naltrexone, the decrease in tics with tramadol was also associated with a significant decrease in OCD symptoms. Although the patient's obsessions continued in an attenuated form, her need to complete her rituals was nearly extinguished. Because of her persistent racing thoughts, divalproex (1500 mg at bedtime) was subsequently initiated with good response. The patient was discharged with a substantially improved condition after a week-long hospitalization. A physician-rated Yale Tourette's Syndrome Symptom List (Revised)[6] showed a score of 32 at baseline and 7 at discharge (where the lower score represents fewer symptoms). Furthermore, 6 months after discharge, Ms. A continues to report good results from tramadol.

Neuroanatomical and neurochemical interactions between basal ganglia dopamine and opioid neurotransmitter systems have recently been described.[7] The striatonigral GABAergic projection neurons express dynorphin, and the prodynorphin gene is positively regulated by D_1-like dopamine receptors. In contrast, the striatopallidal GABAergic neurons express enkephalin, and the preproenkephalin gene is under the tonic inhibitory influence of D_2-like dopamine receptors.[8] It is possible that perturbations of dopaminergic neurotransmission, which may occur spontaneously or which may have been caused by use of dopamine antagonists, such as haloperidol and pimozide, may elicit alterations in the activity of these two distinct opioid peptide systems, which normally are in relative equilibrium. The use of an opioid agonist or antagonist may therefore restore the functional equilibrium within the basal ganglia opioid/dopaminergic systems.

Tramadol may be a safe and effective alternative to current Tourette's syndrome treatments. It is also possible that the reduction this patient experienced in OCD symptoms is related to the simultaneous opioid, noradrenergic, and serotonergic activity of tramadol. Long-term follow-up of this patient and double-blind controlled trials are necessary to assess the effectiveness of tramadol in the treatment of Tourette's syndrome as well as OCD.

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Safety of Amobarbital

Sir: I agree with Dr. Marcum's conclusion that sedative interviews are of questionable value in recovering repressed affects and memories.[1] In fact, prior placebo-controlled studies have shown that intravenous sedatives have minimal value in the anamnesis of the neurotic patient.[2] By comparison, the superiority of a sedative versus placebo in a medication-facilitated interview has been demonstrated in catatonia.[3] Dr. Marcum suggests that midazolam-facilitated interviews should be safer than the less expensive amobarbital since midazolam has a short half-life and its effects could be reversed with flumazenil if necessary. The argument in favor of the greater safety of midazolam would be stronger if there were reports in the *modern* literature of medical complications arising from intravenous amobarbital. I know of none.

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Transient, Paroxysmal, Shock-Like Paresthesias Associated With Paroxetine Initiation

Sir: We report three cases of transient, paroxysmal, shock-like paresthesias associated with the initiation of paroxetine treatment.

Case 1. Ms. A, a 25-year-old Hispanic woman diagnosed by DSM-IV criteria with a major depressive episode and associated anxiety, was placed on paroxetine 20 mg/day. For the first 3 days, shortly after taking her morning 20-mg dose, she experi-